Observations

ON

TYPHOID OR INTESTINAL FEVER: THE PYTHOGENIC THEORY.

BY

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"So the ordinary face and view of experience is many times attested by several theories and philosophies; whereas, to find the real truth, requirerth another manner of severity and attention." *Advancement of Learning.*)

PART III.

DOES INTESTINAL (TYPHOID) FEVER EVER ARISE
SPONTANEOUSLY?

Summary of Conclusions established in preceding Papers. Their general Effect to show Typhoid Fever to be exclusively contagious. The only Evidence to the contrary Negative Evidence. If Typhoid Fever originate spontaneously, by the same Rule Small-pox and Plants and Animals originate in the same Way. First Origin of all these Things wrapped in impenetrable Obscurity. Small-pox. Itch. Syphilis. Resumé of the whole Argument.

THE considerations advanced in preceding papers will probably be regarded as having established the three following propositions:—

1. That intestinal or typhoid fever is an essentially contagious fever.
2. That the largest and most virulent part of the contagious principle is contained in the discharges from the diseased intestine of the fever-patient.
3. That the cloacæ which fill the office of sewer, and which are universally acknowledged to be the principal agents in the propagation of the disease, owe this faculty solely to their being, in the ordinary course of events, the recipients of these discharges; and to the specific infection which they are thus constantly receiving.

But to assert all this is, in effect, to assert that this fever is not only contagious, but exclusively so; or, in other words, that it propagates only by the transmission of its specific germs from the infected to the healthy. For, if it can be shown that even the products of the decomposition of organic matter, for which so strong a case can be made, have in reality no power to cause this disease, there is no single plea for supposing that such a power resides in any other condition.

In reply to this, it has often been objected to me that it is impossible in such a case to prove a negative. But, on the other hand, this alternative is clearly not required. It will be time enough to examine the claims of any other alleged cause when scientific grounds can be produced for advancing them.

In regard to this point, I may further observe that, if this fever really have an origin independent of contagion, facts giving *positive* and express indication of such origin, and incapable of explanation on any other supposition, are sure to make themselves known. Meanwhile, the case admits of being put to a very clear issue. With the exception of what relates to defective sewerage—a case already disposed of—the only plea for believing in the spontaneous origin of this fever consists in evidence of a purely *negative* character. When subjected to a

careful analysis, the whole of this evidence resolves itself into the fact that instances of intestinal fever often spring up in which it is impossible to trace the disease back to a personal source of specific infection. But to this one would have supposed that it would have been sufficient to answer, that this impossibility lies in the very nature of the facts. For, as the fever-poison is invisible to us—its very existence being known to us by inference only—and as, from this and other causes too obvious to need special mention, provision is made for its dissemination in a thousand unseen ways,—it necessarily follows, not only that the precise source of infection in any given case must often escape us, but that cases must *constantly* arise which, to the uninformed, bear all the semblance of spontaneous origin.

The question might fairly be left to rest on these grounds. The violation of all reason involved in the popular mode of solving it may, however, be placed in a still stronger light.

If we are required to believe that intestinal fever originates spontaneously, simply because in many instances we cannot trace the disease back to an immediate prototype, on the very same, or on still stronger grounds, we are bound to believe in the spontaneous origin of small-pox.

In small-pox, indeed, not only do countless cases occur which cannot be traced to contagion, but the disease not rarely springs up under conditions where the idea of such a source would seem at first sight to be altogether shut out. Thus, for example, Dr. Roget saw a man affected with small-pox, who for a considerable time had been under solitary confinement in Millbank Prison.* When small-pox last prevailed extensively in Bristol, it attacked simultaneously three persons who had been for a long while in-patients in three different wards of the Bristol Royal Infirmary. Of these three persons, one had been confined to his bed for more than a month with a fractured leg, another had for some time been altogether disabled, and the third had not left the Infirmary many weeks. What made this example the more telling was that, although there was a good deal of small-pox in Bristol, there was none at the time in the immediate neighbourhood of the house.

Small-pox entirely disappeared from Boston, in New England, seven different times; but in only three of the seven, in spite of the greatest pains taken in the investigation, could the channel of its reintroduction be discovered. (Dr. Henry, *loc. citat.*)

Some years ago, an intimate friend of my own, living in a country house, was seized with small-pox, although he had not been away from home, and although there was, as far as could be ascertained, no other case within forty miles of the place. About twenty years since, an exactly similar instance fell under my own observation in the person of a young boy, in the village of North Tawton, who had never in his life been out of the village. In that case there was certainly no other case of small-pox within a very wide radius of it.

The course of general observation is in entire conformity with these facts.

To those who have not attended to the subject, it

* See for this and other similar instances, an admirable report on Contagion by Dr. Henry of Manchester, in one of the early volumes of the *Transactions of the Provincial Medical and Surgical Association.*

may appear strange, but it is nevertheless strictly true, that since I have lived in Bristol and Clifton—a period covering some twenty years—I have never met with a single instance in private practice in which the patient could trace the disorder to its source. The late Dr. Gregory stated, in his article on Small-pox, in the *Cyclopædia of Practical Medicine*, that of the numerous cases received into the Small-pox Hospital, “not one in twenty is capable of being referred to any known source of infection; the disease being ascribed by the patient to cold, fatigue, change of air, or some other innocent circumstance.”

Judging from my own experience of the two diseases, I can scarcely doubt that, if an accurate register of both were kept in town and country, it would show ten cases of small-pox to one of typhoid fever in which the disease could not be traced to communication with the sick.

From these facts, one of two things is clear: either small-pox, as well as typhoid fever, is constantly springing up anew; or the facts which are commonly supposed in either case to lead to that conclusion offer, in reality, not the slightest warrant for it. There is no possible escape from this dilemma. I need scarcely add that the former alternative will be rejected at once by all whose opinion on the question is worth having.

Dr. Watson has well remarked that the history of small-pox is decisive against the notion of its spontaneous origin. His precise words are these:—“I say the *history* of small-pox leads to the settled belief that this disorder, of which few persons are not readily susceptible, never occurs except from contagion. It was quite unknown in Europe till the beginning of the eighth century. No mention of any such malady is to be found in the Greek or Roman authors of antiquity. Now, whatever may have been the deficiencies of the ancient physicians, they were excellent observers and capital describers of disease; and it is impossible that a disease so diffusive, and marked by characters so definite and conspicuous, should have escaped their notice, or have been obscurely portrayed (if known) in their writings. On the other hand, Mr. Moore, in his learned and interesting *History of Small-pox*, has shown that it prevailed in China and Hindostan from a very early period, even more than a thousand years before the time of our Saviour.”*

If anything were wanting to show what is the true inference to be drawn from these events, it would be found in the fact that, *once imported into the West*, “whensoever or wheresoever the disorder came, it spread with the most fearful rapidity and havoc.” “What I wish you to remark,” says the distinguished writer I have just quoted, “is this: that while almost all men are prone to take the disorder, large portions of the world have remained for centuries exempt from it, until at length it was imported; and that then it infallibly diffused and established itself in those parts.”

* “That it did not sooner extend westward into Persia,” Dr. Watson continues, “and thence into Greece, may be attributed partly to the horror which the disease everywhere inspired, and the attempts that were consequently made to check its progress, by prohibiting all communication with the sick; partly, to the limited intercourse which then took place among the eastern nations, but, principally, to the peculiar situation of the regions through which the infection was diffused; separated, as they were, from the rest of the world by immense deserts, and by the ocean.” (Watson, *Lectures on the Practice of Physic*, 3rd edition, vol. ii, p. 709.)

That the conditions for the manufacture of the poison, if I may so speak, existed here in the most intense degree, was shown by the event when the disease itself was once introduced. The long lapse of ages during which we remained entirely free from it showed with equal clearness that, until this introduction occurred, all those conditions were powerless to cause a single case.*

The spectacle witnessed in Europe was repeated over again in the western world in a still more striking way. Our knowledge of the events here is precise and sure. “There was no small-pox in the New World before its discovery by Columbus in 1492. In 1517, the disease was imported into St. Domingo. Three years later, in one of the Spanish expeditions from Cuba to Mexico, a Negro covered with the pustules of small-pox was landed on the Mexican coast. From him the disease spread with such desolation that within a very short time, according to Robertson, three millions and a half of people were destroyed by it in that kingdom alone.

“Small-pox was introduced into Iceland in 1707, when sixteen thousand persons were carried off by its ravages—more than a fourth part of the whole population of the island. It reached Greenland still later, appearing there for the first time in 1733, and spreading so fatally as almost to depopulate the island.”

What more is needed? Clearly we have here in terms so large that fallacy is out of the question, and so decisive that it is almost impossible even for the dullest or the most perverse to misinterpret them, the history of a specific poison which no common conditions of human life, however various the climate, the people, the civilisation, or however defective the sanitary state, have the power to generate, but for whose propagation the actual virus of a preexisting case is the one necessary and all-sufficient condition. Under this view, everything is at once explained in the most easy and natural manner. Adopt the opposite view, and you are driven at once to the most preposterous assumptions in order to sustain it. This may be made evident by applying the spontaneous theory for a moment to our own case.

When small-pox was first imported into England, it spread, as everywhere else, with the most fearful rapidity. Then, as now, we may be sure—(but, no doubt, to a far greater extent than now, for Jenner had not yet thrown the ægis of vaccination over us)—in addition to those who could trace their disease to direct contagion, vast numbers were attacked who had held no communication with the sick. Then, as now, we may be equally sure, cases were frequently springing up under conditions of the most complete apparent isolation.

If the idea of spontaneous origin be necessary to explain such cases now, it was equally necessary then. So that the assumption we have to make is, that concurrently with the importation of small-pox into England, *but quite independently of that event*, the conditions of English life, which for all time before

* *Ibid.*, p. 710. The same argument is used by Brera, in the following passage. “Il vajuolo pare che talvolta sorprenda degli individui che non ebbero comunicazione co' vajuolosi. Eppure egli è deciso, che il vajuolo era, sono undici secoli, affatto sconosciuto in Europa, e che è rimasto sconosciuto in alcune isole del mar pacifico, fino a tanto che non vi penetrarono gli Europei.” (Brera, *De Contagi*, vol. i, p. 102.)

had been powerless to develop a single case, had suddenly acquired the power to breed the disease in the most prolific manner.

I need scarcely add that the bare statement of a conclusion such as this is the best exposure of its extravagant absurdity.

Our difficulties, insuperable as they already are, would not indeed end here. For, even if all this were granted, we should further have to explain how it is that the English people, having once acquired so perfectly the art of brewing this specific poison, should by migrating to Australia and New Zealand, have again altogether lost it. Happily, as yet (although in Australia, at any rate, the conditions of epidemic disease are ripe enough), this particular scourge has not once made its appearance in either of these two colonies.

I may add, that the philosopher probably does not exist who would not readily hazard the prediction that, if by neglect or mischance small-pox should be imported into these communities, they would at once appear to have recovered the power of breeding it; and observers with more zeal than knowledge would there, as here, again point to isolated examples of it as proofs of its spontaneous origin.

[To be continued.]

Original Communications.

ON RINGWORM AND VEGETABLE PARASITES.

By THOMAS HILLIER, M.D. Lond., Assistant-Physician to the Hospital for Sick Children.

[Read before the North London Medical Society.]

[Concluded from page 554.]

MR. HOGG maintains that parasitic vegetation is not the cause, but the result of disease, on the following grounds:—"1. The general law that vegetable parasites only attack bodies in a state of lowered vitality." "2. The growth of fungi is not necessarily pathognomic of a special disease, because they have been observed in all kinds of chronic skin-diseases." Mr. Hogg says he has discovered a fungus in psoriasis, lepra, eczema, spilus, ichthyosis, and lichen. "3. Competent observers have not been able to find them in the diseases which they are believed to engender." "4. As to the results of inoculation, the experiments are too few and irregular to afford any basis for argument *pro* or *con*." (*Trans. Microsc. Journ.*, 1859.)

In reply to these statements, it may be said that, in tinea tonsurans and tinea favosa, at any rate, all competent observers find a fungus, with the exception of Mr. Erasmus Wilson, who sees the phenomena, however, but interprets them differently. In the chronic diseases to which Mr. Hogg refers, if a fungus sometimes exist, its presence must be regarded as accidental, and its extent not at all proportionate to the amount of disease, being entirely absent even in aggravated cases of these affections. I have searched in vain for a fungus in several cases of psoriasis and lichen. But in those diseases in which the fungus is never absent, and where the changes produced by the microscope are those most obvious both to the naked eye and to the microscope, we may certainly give the parasite a more prominent place in the pathology of the disease; and it is only reasonable to regard it as the determining cause of the disease, whilst we admit constitutional and local states as predisposing causes.

Tinea tonsurans and tinea favosa cannot exist without their respective fungi. Pityriasis versicolor is always accompanied by a fungus; but pityriasis rubra and pityriasis capitis exist usually, if not always, without a fungus. Herpes circinatus is commonly accompanied by a fungus, and probably some cases are due to the fungus; whilst other cases of this affection are found without the fungus; so that we cannot say positively that it is always a parasitic disease. Sycosis probably is always parasitic in its origin. In reference to alopecia areata or tinea decalvans, some observers, including MM. Gruby, Bazin, Hogg, and Lowe, discover a fungus which has been called *microsporon Audouini*. Dr. Jenner and Mr. Hutchinson fail to detect it, and I have been equally unsuccessful. That a fungus sometimes attacks the hairs in these cases, must be admitted on the authority of such observers as those mentioned; but we must also maintain that it is frequently absent; so that I would class this disease with the non-parasitic diseases, in which a fungus is occasionally found, but does not determine the existence of the disease.

The evidence derived from treatment is very strong in favour of the parasitic theory, although it must be admitted that this argument applies partially in reference to some diseases where no parasite has been detected; namely, porrigo larvaris, or the contagious impetigo of the scalp. Alopecia areata, too, will generally yield to solution of sulphurous acid. This may, however, be due to its irritant properties, and not to its power of destroying vegetable life; as blistering is more efficacious than the sulphurous lotion.

Parasitocides will, I believe, in all cases cure tinea favosa, tinea tonsurans, sycosis, and pityriasis versicolor. The length of time required to cure tinea tonsurans is owing to the impossibility of getting the parasitocides to the bottom of the hair-follicles in which the fungus has embedded itself. At the very outset, it may be cured rapidly. Newly attacked parts, before the hairs were much attacked, in many of my cases, were cured in a few days.

Dr. Lowe (*Botan. Trans.*, Edin., 1858) endeavours to prove an identity between the fungus in tinea favosa and the *aspergillus glaucus*. He is inclined to concur with Dr. Fox as to the common origin of all the human fungi. He says that *trichophyton tonsurans* is only the sporular form of *achorion Schönleini*. He placed a mass of cells from favus in a solution of brown sugar, and at the end of five weeks he found complete plants of *aspergillus glaucus*, with the fructification in its characteristic form. Remak, who made similar experiments on the development of the achorion, obtained no results from favus-sporules placed in a saccharine solution, but found that those placed on apple germinated and put out processes in twenty-four hours; but on the sixth day the apple was covered with *penicillium glaucum*. He says: "At this time, the surface of the apple changed its colour in a surprising manner to brown; there appeared on the entire surface a rapid growth of *penicillium glaucum* or other kinds of mould, which very much interfered with the distinguishing of those fungi which belonged to the favus from those which belonged to other kinds of mould that has sprung up from unobserved sporules." He also states, that on the fourth or fifth day he had observed in the interior of the piece of crust a powdery substance evidently arising from a decomposition of this favus-fungus. In the saccharine solution he found the crust of favus "take the form merely of slender filaments of mycelium; and by the side of it a powdery mass, which probably owed its origin to the decomposed parts of the favus-fungus." (*Diagnost. und pathogenetische Untersuchungen*, Berlin, 1845, pp. 200-2.) Dr. Lowe thinks that the favus in Remak's experiments originated the *penicillium*; and that it, as well as *aspergillus*, is capable of originating the disease.